test to compare the outcome of various management strategies in each CAE type.
Results: 2539 Coronary angiograms were done in our hospital. The incidence of CAE is 1.22% (N=31).74.2% were male. The mean age, body surface area, oxygen consumption and Low Density Lipoprotein levels were 61.23±9.8years, 1.7±0.14m^2, 202.9±33ml/min and 83.8±27.6mg/dl. No significant association with diabetes mellitus or hypertension but negative association with smoking [Likelihood ratio 0.032]. 77.5% of CAE had left ventricle systolic function >40%. 13 (41.9%) patients underwent CABG, 12 (38.7%) were on medical management and 5(16.1%) had PCI. One patient had dilatation of ascending aorta along with LAD ectasia and underwent Bentalls. Type I (50%, N=5) & IV (50%, N=5) lesion were predominantly underwent CABG. Whereas both type II (50%, N=2) & III (42.9%, N=3) were kept on medical management. PCI was mostly the option for type III (40%, N=2) ectasia. Six months follow up showed no events in any of the patients.
Conclusions: CAE has got over all good prognoses. There is no association of diabetes or hypertension with Ectasia. But there is negative association of smoking with Ectasia. Type II & III ectasia can be put on medical management alone. Type I ectasia require CABG.

Assessment of high sensitivity C-reactive protein in ETT positive patients with normal coronary angiogram

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Introduction: About 30% of coronary angiographies done for chest discomfort and positive stress cardiac testing are normal. Patients with chest pain with normal coronary arteries have coronary microvascular endothelial dysfunction and myocardial ischemia. Elevated hs-CRP levels have been related to atherogenesis and endothelial dysfunction. Little is known whether low grade chronic inflammation is a pathogenic mechanism.
Aims and objectives: To assess high sensitive CRP in patients of typical chest pain with normal coronary arteries (cardiac Syndrome X).
Methods: Cardiac Syndrome - X patients were compared to controls to see any difference of markers of inflammation in the form of HS-CRP. 120 patients with 50 number of well matched controls were studied. All the patients underwent baseline investigations, ECG, ETT , Echocardiography and coronary angiographies. The serum levels of hs-CRP were estimated.
Results: Among the study group (Group-1), the mean age was 48.12 (± 7.87) yrs and 47.48 (± 7.48) yrs among control group (Groyp-2). In Group-1 , 96 (80%) were male and 24 (20%) were female. In Group-2 , 40 (80%) were male and 10 (20%) were female . In Group-1, 60% had sedentary lifestyle, 60% were hypertensives, and 50% were diabetics or IGT, 70% were smokers, 40% were dyslipidemics, 30% had family history of CAD and 50% were obese. and serum levels of hs-CRP were found to be significantly higher in Group-1 than in Group-2 patients, (4.10 ± 2.74 mg/L vs 1.18 ± 0.9 6mg/L, $p < 0.001$).
Conclusion: hs-CRP levels are higher in patients of cardiac Syndrome-X, suggesting a chronic low grade inflammatory process.

Association of coronary artery disease and carotid artery disease in patients with peripheral vascular disease

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Background: In the decade ahead, patients and primary care physicians will increasingly recognize the clinical burden of peripheral arterial disease (PAD). As new advances in the treatment of coronary artery disease continue to reduce mortality and morbidity, caregivers will increasingly confront the problem of concomitant "noncoronary" arterial disease. Cardiovascular physicians should assume a more proactive clinical role, along with their vascular medicine colleagues, to encourage new therapeutic opportunities for the treatment of arterial disease affecting multiple vascular beds. Multivascular therapeutic approaches are needed because atherosclerosis has a common systemic pathogenesis and simultaneously affects multiple circulations.
Objectives: Peripheral vascular disease patients are at high risk of developing cardiovascular and cerebrovascular events. The aim of this study was to see the association of Coronary artery disease and Carotid artery disease in patients with Peripheral vascular disease.
Methods: 2 50 (200 males and 50 females) patients with peripheral vascular disease of lower limbs admitted in the National Institute of Cardiovascular Diseases (NICVD), Dhaka between June 2012 to July 2013. Coronary angiography and carotid Doppler study was done in all patients during their hospital stay.
Results: 125 of 250 patients (50 %) had significant CAD and 50 patients (20%) had significant carotid artery lesion and 25 patients (10%) had both. In patients with significant CAD, 75 patients of 125 (60%) had severe CAD (left main or triple vessel or proximal LAD lesion). 13 patients (10.5%) had left main, 50 patients (40%) had triple vessel disease and 12 patients (09.5%) had proximal LAD lesion . Among the 25 patients with both lesion, 15 patients (60%) had severe CAD of which 3 patient (12%) had left main, 4 patients (16%) had triple vessel disease and 3 patients (12%) had proximal LAD lesion. The percent of patients with severe CAD (left main, 3 vessel or proximal left anterior descending lesion) among those with Carotid artery disease was higher compared to those without Carotid artery disease.
Conclusions: Both or either Coronary artery disease and/ or Carotid artery disease are quite prevalent in patients with Peripheral vascular disease. So, all peripheral arterial disease patients should be investigated to see presence of any coronary artery disease or carotid artery disease.

Neutrophil count and its correlation with short term morbidity and mortality in patients with acute coronary syndrome

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Background: Inflammation plays a key role in the development of atherosclerosis and acute coronary syndromes, the most important cause of sudden cardiac death. Certain inflammatory

markers have been identified to be associated with increased incidence of cardiovascular complications and death in patients with ACS and increased severity of coronary artery lesion. Among these biomarkers some have emerged to be potentially useful in early risk assessment of patients with ACS, such as CRP, B type natriuretic peptide, troponin T/I, interleukin 6, and white blood cell (WBC) count and among these WBC count is the simplest. Although total leukocyte count is an established prognostic marker of ACS, there are limited studies for differential neutrophil count and prognosis in ACS.

Methods: 202 consecutive patients admitted with a diagnosis of ACS were evaluated by history and physical examination. Venous blood was drawn from all the patients at the time of admission and was analysed for CK-MB, Troponin T and Total leukocyte count and differential leukocyte count. The neutrophil counts were assigned into three categories, N1 (<70%), N2 (70-90%) and N3 (≥90%): Coronary angiography was carried out within 24 hours of admission.

Results: The Neutrophil count ranged from 45% to 98%. The median neutrophil count was 78%; Smokers had a higher neutrophil count than non-smokers. Neutrophil count also tended to be higher in patients who died within 7 days (P<0.001). The development of new CHF or shock was associated with a higher Neutrophil count (P<0.001) Patients with a closed infarct-related artery on angiography (TIMI grade 0 or 1 flow) had a higher neutrophil count than did patients with an open artery (P<0.001). The presence of angiographically apparent thrombus was associated with a higher Neutrophil count than those without thrombus. (82%, n=28 versus 68%, n=72) (p<.01)

Conclusion: The results of the present study confirm previous observations that relate elevated WBC count to adverse clinical outcomes in patients with ACS and further explore the pathophysiology that underlies this relationship. In addition to the worse clinical outcome, reduced patency and greater thrombus burden seen in patients with an elevated Neutrophil count, these patients had poorer downstream microvascular perfusion as assessed with TIMI perfusion grade. It is possible that this impaired myocardial perfusion reflects neutrophil–mediated endothelial dysfunction and microvascular plugging, as described in animal models of ischemia-reperfusion.

Single nucleotide polymorphisms associated with myocardial infarction in patients from Western India: A genome wide association study

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Background: Myocardial infarction (MI) is leading cause of death worldwide. Moreover, in India the average age of acute myocardial infarction (AMI) has reduced to 50 indicating it as a grave public health concern. The objective of our study was to identify genomic variants associated with AMI in patients from Western India in genome wide association study (GWAS) and validating the identified single nucleotide polymorphisms (SNPs), using high-throughput DNA microarray analysis.

Methods: Initially, 48 AMI patients and 48 controls were screened for SNPs using Illumina human CVD55K beadchip, containing approximately 50,000 human SNPs probes. The identified SNPs were then further validated by genotyping additional 188 patients and 196 controls using custom based Illumina's VeraCode GoldenGate Genotyping Assay. PLINK software was used to perform statistical analysis.

Results: On normalization and filtration of the preliminary microarray data, 98 SNPs of 94 genes were identified associated with AMI (odds ratio range of 1.84-8.85, p value 0.0486 to 0.00334). Eight of these 98 SNPs reproduced association (p<0.05). The genes associated with some SNPs encoded for the proteins linked with blood coagulation, innate immunity, troponin complex and inflammatory pathways.

Conclusion: The study identified 8 SNPs associated with AMI which may increase the susceptibility towards the disease in patients from Western India.

T peak – T end interval: Marker for arrhythmic events at 30 days following ST elevation myocardial infarction

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Objectives: We aimed to analyze the effect of reperfusion of infarct related artery on the TpTe interval determined on the surface 12 lead ECG. We also studied the association of Major adverse cardiac events (MACE) with repolarization abnormality on the ECG.

Methods: Patients with new onset STEMI treated with thrombolysis or primary/ rescue PCI were included. Digital ECGs at 50 mm/sec speed and 20 mm/mV gain filtered at 0.50–150Hz were taken before and after reperfusion therapy. TpTe interval was measured in leads with limited ST-segment deviation. All patients were followed up at 30 days.

Results: From June 2013 to December 2013, total of 216 patients were included of which 183 were males (85.1%). The mean age was 54.86 years (range 24-80 years). One hundred and thirteen patients underwent primary PCI (52.3%), 57 lysis (26.4%) and remaining 46(21.3%) rescue PCI. Thirty day Mortality was 5.1 % (11 patients). The pre TpTe interval was 84.50ms (IQR 80 - 100 ms) and the mean TpTe intervals reduced following intervention; primary PCI (74.93ms, p <0.001), thrombolysis (72.76, p<0.001) and rescue PCI groups (72.86 ms, p=0.004). Of the 216 patients, 210 were followed up at 30 days. Six patients were lost to follow up. Eleven patients died 11(5.1%) patients died. Pre TpTe interval more than 100 ms predicted (OR 13.21, 95% CI 1.16 – 150.57) increased risk of arrhythmias (Ventricular Tachycardia and Ventricular Fibrillation). However, it did not predict mortality at 30 days (OR – 1.4, 95% CI – 0.28 – 6.84). After adjusting for established risk factors, TpTe interval difference (pre – post) was found to be significantly associated with duration of chest pain and Killip class.

Conclusion: The TpTe interval was significantly reduced after reperfusion therapy. Pre-reperfusion TpTe predicted the risk of arrhythmias at 30 days. However, it did not predict subsequent all-cause mortality and heart failure at 30 days.